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FILE COVERS 1907 - 6 May 2003 VOL 138 ISS 19
FILE LAST UPDATED: 5 May 2003 (20030505/ED)

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L3 180 SEA FILE=REGISTRY CLA/BI
L5 281162 SEA FILE=HCAPLUS L2 OR L3 OR CONJUGATED(W)LINOLEIC(W)ACID? OR
CLA
L10 (52)SEA FILE=REGISTRY INDOMETHACIN/BI
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L14 (3)SEA FILE=REGISTRY 3,4-DIHYDROXYCINNAMIC ACID?/CN
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L16 (68)SEA FILE=REGISTRY GOSSYPOL/BI
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L18 (0)SEA FILE=REGISTRY BACOMYCESIC ACID?/CN
L19 (82)SEA FILE=REGISTRY 3,4-DIHYDROXYPHENYL(L)ETHANOL?
L20 (180)SEA FILE=REGISTRY CLA/BI
L21 388823 SEA FILE=HCAPLUS L10 OR L11 OR L12 OR L13 OR L14 OR L15 OR L16
OR L17 OR L18 OR L19 OR L20
L22 3958 SEA FILE=HCAPLUS L1 (L)(INHIB? OR REDUC? OR ACTIV? OR ADIPO?
OR OBES? OR BODY(W)FAT OR FAT(W)BODY)
L23 384 SEA FILE=HCAPLUS L21 AND L22
L44 7 SEA FILE=HCAPLUS L23 AND L5

=> d ibib abs hitrn 144 1-7

L44 ANSWER 1 OF 7 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:978341 HCAPLUS
DOCUMENT NUMBER: 138:33341
TITLE: Animal body fat control by reducing lipoxxygenase
activity
INVENTOR(S): Pariza, Michael W.; Park, Yeonhwa
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 13 pp.

Searched by M. Smith

DOCUMENT TYPE: CODEN: USXXCO
LANGUAGE: Patent
FAMILY ACC. NUM. COUNT: English
PATENT INFORMATION: 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002197301	A1	20021226	US 2001-871125	20010531
PRIORITY APPLN. INFO.:			US 2001-871125	20010531

AB A method for controlling body fat in a human or nonhuman animal includes the step of reducing lipoxxygenase activity in an animal. Lipoxxygenase activity can be reduced by reducing the enzyme activity or by lowering the enzyme level. Reduced lipoxxygenase activity correlates with reduced cell-assocd. lipoprotein lipase (LPL) activity and with reduced cellular triacylglyceride level. Mice fed feed contg. lipoxxygenase inhibitor nordihydroguaiaretic acid (NDGA) at a 0.1 % level had reduced fat and increased water and protein. A synergistic effect on body fat compn. was seen with a combination of NDGA and **conjugated linoleic acid** at 0.1 %.

IT **63551-74-6, Lipoxxygenase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(animal **body fat** control by **reducing**
lipoxxygenase **activity**)

IT **1839-11-8, Conjugated linoleic acid**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
(animal body fat control by reducing lipoxxygenase activity)

IT **53-86-1, Indomethacin 89-73-6, Salicylhydroxamic acid**
303-45-7, Gossypol 331-39-5, 3,4-Dihydroxycinnamic acid
491-67-8, Baicalein 1191-85-1, ETYA 10597-60-1
, 3,4-Dihydroxyphenyl ethanol 58688-54-3, 5,6-Dehydro
arachidonic acid
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
(lipoxxygenase inhibitor; animal body fat control by reducing
lipoxxygenase activity)

L44 ANSWER 2 OF 7 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:56455 HCAPLUS

DOCUMENT NUMBER: 136:319029

TITLE: Lipoxxygenase inhibitors inhibit heparin-releasable
lipoprotein lipase activity in 3T3-L1 adipocytes and
enhance body fat reduction in mice by
conjugated linoleic acid

AUTHOR(S): Park, Yeonhwa; Pariza, Michael W.

CORPORATE SOURCE: Food Research Institute, Department of Food
Microbiology and Toxicology, University of
Wisconsin-Madison, Madison, WI, 53706, USA

SOURCE: Biochimica et Biophysica Acta (2001), 1534(1), 27-33
CODEN: BBACAQ; ISSN: 0006-3002

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The t10c12 isomer of **conjugated linoleic acid**
(**CLA**) reduces lipid accumulation in adipocytes in part by
inhibiting heparin-releasable lipoprotein lipase (LPL) activity. We now
show that inhibitors of lipoxxygenase (LOX) activity (2-[12-hydroxydodeca-
5,10-diynyl]-3,5,6-trimethyl-p-benzoquinone; 5,8,11,14-eicosatetraynoic

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L44 ANSWER 1 OF 7 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:978341 HCAPLUS

DOCUMENT NUMBER: 138:33341

TITLE: Animal body fat control by reducing lipoxxygenase
activity

INVENTOR(S): Pariza, Michael W.; Park, Yeonhwa

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 13 pp.

Searched by M. Smith

acid; salicylhydroxamic acid; indomethacin; nordihydroguaiaretic acid (NDGA)) produce a similar inhibitory effect on LPL activity in cultured 3T3-L1 mouse adipocytes. Addnl. the LOX inhibitors had no effect on, or inhibited, lipolysis in this cell system (measured as glycerol release). Growing mice fed diet contg. 0.1% NDGA for 4 wk displayed 21% redn. in body fat, which was similar to 23% redn. in body fat produced by feeding diet contg. a suboptimal amt. of **CLA** (0.1%) for 4 wk. Feeding diet contg. both 0.1% NDGA and 0.1% **CLA** resulted in 51% redn. in body fat which was accompanied by significant increases in whole body water and protein. Aspirin, an inhibitor of cyclooxygenase 1 and 2, had no effect on LPL activity in 3T3-L1 adipocytes, did not affect body compn. when fed to growing mice, and failed to influence the effects of **CLA** on LPL activity in 3T3-L1 cells or body compn. in mice. These findings appear to provide new perspectives and insights into the relationships between **CLA**, eicosanoids, the control of lipid accumulation in adipocytes, and effects of **CLA** on the immune system.

IT 9029-60-1, Lipoxygenase

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(lipoxygenase **inhibitors inhibit** heparin-releasable lipoprotein lipase **activity** in 3T3-L1 **adipocytes** and enhance **body fat** redn. in mice by **conjugated linoleic acid**)

IT 53-86-1, Indomethacin 89-73-6, Salicylhydroxamic acid

1191-85-1, 5,8,11,14-Eicosatetraynoic acid 121250-47-3,
Conjugated linoleic acid

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(lipoxygenase inhibitors inhibit heparin-releasable lipoprotein lipase activity in 3T3-L1 adipocytes and enhance body fat redn. in mice by **conjugated linoleic acid**)

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 3 OF 7 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2000:688272 HCAPLUS

DOCUMENT NUMBER: 133:280563

TITLE: Human antibodies that bind human IL-12 and methods for producing

INVENTOR(S): Salfeld, Jochen G.; Roguska, Michael; Paskind, Michael; Banerjee, Subhashis; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra; Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela; Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela; Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara; Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L.

PATENT ASSIGNEE(S): Basf A.-G., Germany; Genetics Institute Inc.; et al.

SOURCE: PCT Int. Appl., 377 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000056772	A1	20000928	WO 2000-US7946	20000324
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR,				

CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU,
 ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU,
 LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE,
 SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA,
 ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
 RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
 DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
 CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
 NZ 513945 A 20010928 NZ 2000-513945 20000324
 BR 2000009323 A 20020108 BR 2000-9323 20000324
 EP 1175446 A1 20020130 EP 2000-918396 20000324
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, SI, LT, LV, FI, RO
 JP 2002542770 T2 20021217 JP 2000-606632 20000324
 NO 2001004605 A 20011126 NO 2001-4605 20010921
 PRIORITY APPLN. INFO.: US 1999-126603P P 19990325
 WO 2000-US7946 W 20000324

AB Human antibodies, preferably recombinant human antibodies, that
 specifically bind to human interleukin-12 (hIL-12) are disclosed.
 Preferred antibodies have high affinity for hIL-12 and neutralize hIL-12
 activity in vitro and in vivo . An antibody of the invention can be a
 full-length antibody or an antigen-binding portion thereof. The
 antibodies, or antibody portions, of the invention are useful for
 detecting hIL-12 and for inhibiting hIL-12 activity, e.g., in a human
 subject suffering from a disorder in which hIL-12 activity is detrimental.
 Nucleic acids, vectors and host cells for expressing the recombinant human
 antibodies of the invention, and methods of synthesizing the recombinant
 human antibodies, are also encompassed by the invention.

IT 9029-60-1, Lipoxxygenase
 RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL
 (Biological study); USES (Uses)
 (inhibitors; recombinant human antibodies that bind human
 IL-12 for treatment of autoimmune diseases and inflammatory diseases)

IT 50-18-0, Cyclophosphamide
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (recombinant human antibodies that bind human IL-12 for treatment of
 autoimmune diseases and inflammatory diseases)

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS
 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 4 OF 7 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1999:48609 HCAPLUS

DOCUMENT NUMBER: 130:119591

TITLE: Antioxidant enhancement of therapy for
 hyperproliferative conditions

INVENTOR(S): Chinery, Rebecca; Beauchamp, R. Daniel; Coffey, Robert
 J.; Medford, Russell M.; Wadsinski, Brian

PATENT ASSIGNEE(S): Atherogenics, Inc., USA

SOURCE: PCT Int. Appl., 112 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
WO 9901118	A2	19990114	WO 1998-US13750	19980701
WO 9901118	A3	19990422		

W: AL, AU, BA, BB, BG, BR, CA, CN, CU, CZ, EE, GE, HU, ID, IL, IS, JP, KP, KR, LC, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL, RO, SG, SI, SK, SL, TR, TT, UA, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9882827 A1 19990125 AU 1998-82827 19980701
 EP 1019034 A2 20000719 EP 1998-933078 19980701

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

JP 2002511878 T2 20020416 JP 1999-507360 19980701
 US 2001049349 A1 20011206 US 2001-779086 20010207

PRIORITY APPLN. INFO.: US 1997-886653 A 19970701
 US 1997-967492 A 19971111
 US 1998-108609 B1 19980701
 WO 1998-US13750 W 19980701

OTHER SOURCE(S): MARPAT 130:119591

AB A method to enhance the cytotoxic activity of an antineoplastic drug comprises administering an effective amt. of the antineoplastic drug to a host exhibiting abnormal cell proliferation in combination with an effective cytotoxicity-increasing amt. of an antioxidant. The invention also includes a method to decrease the toxicity to an antineoplastic agent or increase the therapeutic index of an antineoplastic agent administered for the treatment of a solid growth of abnormally proliferating cells, comprising administering an antioxidant prior to, with, or following the antineoplastic treatment.

IT 50-18-0, Cyclophosphamide
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(antioxidant enhancement of therapy for hyperproliferative conditions)

IT 9029-60-1, Lipoxigenase
 RL: BSU (Biological study, unclassified); BIOL (Biological study) (inhibitors; antioxidant enhancement of therapy for hyperproliferative conditions)

L44 ANSWER 5 OF 7 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1994:569933 HCAPLUS

DOCUMENT NUMBER: 121:169933

TITLE: Cyclooxygenase and lipoxigenase inhibitors as modulators of cancer therapies

AUTHOR(S): Teicher, Beverly A.; Korb, Timothy T.; Menon, Krishna; Holden, Sylvia A.; Ara, Gulshan

CORPORATE SOURCE: Dana-Farber Cancer Inst., Boston, MA, 02115, USA
 SOURCE: Cancer Chemotherapy and Pharmacology (1994), 33(6), 515-22

CODEN: CCPHDZ; ISSN: 0344-5704

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Like many clin. non-small-cell lung cancers, the Lewis lung carcinoma produces prostaglandins. The Lewis lung carcinoma was used as a model of both primary and metastatic disease to assess the ability of cyclooxygenase inhibitors (mefenamic acid, diflunisal, sulindac, and indomethacin), the collagenase inhibitor minocycline, and the lipoxigenase inhibitor phenidone to act as modulators of cytotoxic cancer therapies. Although none of the single modulators given i.p. daily on days 4-18 altered tumor growth or the no. of metastases found on day 20, modulator combinations consisting of minocycline/a cyclooxygenase inhibitor and,

esp., of phenidone/a cyclooxygenase inhibitor resulted in modest tumor growth delay and a decreased no. of lung metastases on day 20. The most effective modulators of cisplatin (CDDP) were phenidone/sulindac and phenidone/indomethacin, which led to 2.4- to 2.5-fold increases in the tumor growth delay produced by CDDP. The most effective modulations of cyclophosphamide resulted from administration of minocycline, minocycline/sulindac, or phenidone/sulindac and led to 2.0- to 2.1-fold increases in tumor growth delay by cyclophosphamide. The most effective modulators of melphalan produced 4.5- to 4.7-fold increases in tumor growth delay by the drug and were minocycline/sulindac, minocycline/mefenamic acid, and phenidone/sulindac. The most effective modulation of carmustine (BCNU) was obtained with minocycline/sulindac and minocycline/diflunisal leading to 2.8- to 3.1-fold increases in tumor growth delay by BCNU. Finally, the most effective modulation of radiation was obtained with minocycline/sulindac and phenidone/sulindac and resulted in 2.8- to 3.3-fold increases in tumor growth delay by radiation. The modulator combination that along with the cytotoxic therapies was most effective against metastatic disease was phenidone/mefenamic acid. There was no clear relationship between effective modulation of the cancer therapies and the degree of redn. in serum levels of prostaglandin E2 and leukotriene B4 by the agents in Lewis lung tumor bearing mice.

- IT 53-86-1, Indomethacin
 RL: BIOL (Biological study)
 (antitumor drugs modulation by lipoxxygenase inhibitors and, as cyclooxygenase inhibitor)
- IT 50-18-0, Cyclophosphamide
 RL: PRP (Properties)
 (antitumor effect of, cyclooxygenase and lipoxxygenase inhibitors modulation of)
- IT 63551-74-6
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (inhibitors, antitumor drugs modulation by cyclooxygenase inhibitors and)

L44 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1989:433265 HCAPLUS

DOCUMENT NUMBER: 111:33265

TITLE: Effects of anti-inflammatory drugs on interleukin 1-induced cartilage proteoglycan resorption in vitro: inhibition by aurothiophosphines but no influence from perturbed eicosanoid metabolism

AUTHOR(S): Rainsford, K. D.

CORPORATE SOURCE: Anti-Inflammatory Res. Unit, Strangeways Res. Lab., Cambridge, CB1 4RN, UK

SOURCE: Journal of Pharmacy and Pharmacology (1989), 41(2), 112-17

CODEN: JPPMAB; ISSN: 0022-3573

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A range of anti-inflammatory drugs having varying effects on eicosanoid metab. and other actions was studied for their potential to inhibit .alpha.-interleukin I (IL-1)-induced cartilage proteoglycan resorption in vitro. No effects on resorption were obsd. with inhibitors of cyclooxygenase or lipoxxygenase or mixed inhibitors of both these enzymes, and no influence on IL-1 effects was obsd. with added eicosanoids. Among the clin. used disease-modifying antiarthritic agents, only auranofin and the immunoregulatory agent tilomisole were effective in inhibiting resorption. Some auranofin analogs having Cl or NO2 leaving groups that inhibit DNA polymerase-.alpha. were potent inhibitors of IL-1 induced

- resorption.
- IT 80619-02-9, 5-Lipoxygenase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors, resorption of proteoglycans of cartilage response to)
- IT 50-18-0, Cyclophosphamide
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(resorption of proteoglycans of cartilage response to)

L44 ANSWER 7 OF 7 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1986:102126 HCAPLUS
DOCUMENT NUMBER: 104:102126
TITLE: Modulation of mouse ear edema by cyclooxygenase and lipoxygenase inhibitors and other pharmacologic agents
AUTHOR(S): Carlson, Richard P.; O'Neill-Davis, Lynn; Chang, Joseph; Lewis, Alan J.
CORPORATE SOURCE: Dep. Exp. Therapeut., Wyeth Lab., Inc., Philadelphia, PA, 19101, USA
SOURCE: Agents and Actions (1985), 17(2), 197-204
CODEN: AGACBH; ISSN: 0065-4299
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Inhibitors** of arachidonic acid (AA) [506-32-1] metab. and other pharmacol. agents were evaluated against ear edema produced in mice by tetradecanoylphorbol acetate (TPA) or AA. Drugs were administered orally and topically either 30 min prior to AA or 30 min after TPA, except for steroids which were administered 2.5-3 h prior to AA. Several cyclo-oxygenase [39391-18-9] (CO) **inhibitors** including indomethacin [53-86-1], aspirin [50-78-2], piroxicam [36322-90-4] and timegadine [71079-19-1] were without effect when administered orally against either irritant; the same drugs **inhibited** TPA edema when they were administered topically. Mixed CO/lipoxygenase (LO) [9029-60-1] **inhibitors**, phenidone [92-43-3] and BW755C [66000-40-6], were **active** orally against AA edema (ED50s of 84 and 65 mg/kg, resp.) and against TPA edema (ED50s of 235 and 88 mg/kg, resp.). Phenidone was more **active** topically against AA edema (ED50, 0.1 mg/ear) than BW755C (ED50, 2.8 mg/ear); however, BW755C was more **active** topically against TPA edema (ED50, 0.2 mg/ear) than phenidone (ED50, 0.6 mg/ear). Methylprednisolone [83-43-2] was very effective in the AA (oral ED50, 17 mg/kg; topical ED50, .degree.1 mg/ear) and TPA models (oral ED50, 4.3 mg/kg; topical ED50, 0.03 mg/ear). MK-447 [58456-91-0] was topically and orally effective only in the TPA model. Not surprisingly, drugs were more effective topically than orally in both mouse ear edema assays. The models were somewhat selective for CO and CO/LO **inhibitors**; however, dapsone [80-08-0] was orally effective in the ear models, and a no. of mediator antagonists and central nervous system drugs, esp. antipsychotics, were topically **active** primarily against TPA edema. These models may be useful for the detection of in vivo **activity** of CO/LO or 5-LO **inhibitors**.

- IT 50-18-0 53-86-1
RL: PRP (Properties)
(anti-inflammatory effect of, in ear edema)
- IT 9029-60-1
RL: BIOL (Biological study)
(**inhibitors** of, anti-inflammatory effect of, in ear edema)

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        OR OBES? OR BODY(W)FAT OR FAT(W)BODY)
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L34 (  29803)SEA FILE=HCAPLUS L25 OR INDOMETHACIN?
L35 (   567)SEA FILE=HCAPLUS L26 OR ETYA
L36 (  1522)SEA FILE=HCAPLUS L27 OR SHA
L37 ( 1193)SEA FILE=HCAPLUS L28 OR BAICALEIN?
L38 (   268)SEA FILE=HCAPLUS L29 OR 3(W)4(W)DIHYDROXYCINNAMIC(W)ACID?
L39 (   18)SEA FILE=HCAPLUS CINNAMYL(L)3(W)4(W)DIHYDROXY(L)CYANOCINNAMATE?

L40 (   3381)SEA FILE=HCAPLUS L30 OR GOSSYPOL
L41 (    25)SEA FILE=HCAPLUS L31 OR 5(W)6(W)DEHYDRO(W)ARACHIDONIC(W)ACID
L42 (    23)SEA FILE=HCAPLUS L32 OR BAEOMYCESIC(W)ACID?
L43     36495 SEA FILE=HCAPLUS (L33 OR L34 OR L35 OR L36 OR L37 OR L38 OR
        L39 OR L40 OR L41 OR L42)
L44      7 SEA FILE=HCAPLUS L23 AND L5
L45     606 SEA FILE=HCAPLUS L22 AND L43
L46      5 SEA FILE=HCAPLUS L45 AND L5
L47      1 SEA FILE=HCAPLUS L46 NOT L44

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=> d ibib abs hitrn 147

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L47 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:198591 HCAPLUS
DOCUMENT NUMBER: 126:258607
TITLE: Proliferation responses of normal human mammary and
        MCF-7 breast cancer cells to linoleic acid,
        conjugated linoleic acid
        and eicosanoid synthesis inhibitors in culture

```

AUTHOR(S): Cunningham, Diane C.; Harrison, Lisa Y.; Shultz, Terry D.
 CORPORATE SOURCE: Dep. Food Sci. Human Nutrition, Washington State Univ., Pullman, WA, 99164-6376, USA
 SOURCE: Anticancer Research (1997), 17(1A), 197-203
 CODEN: ANTRD4; ISSN: 0250-7005
 PUBLISHER: Anticancer Research
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB Potential mechanisms for the stimulation or inhibition of cell growth by linoleic acid (LA) and **conjugated linoleic acid (CLA)** were investigated by using eicosanoid synthesis inhibitors. Normal human mammary epithelial cells (HMEC) and MCF-7 breast cancer cells were incubated in serum-free medium supplemented with LA or **CLA** and cyclooxygenase (**indomethacin**; INDO) or lipoxygenase (nondihydroguaiaritic acid; NDGA) inhibitors. Linoleic acid stimulated the growth and [3H]thymidine incorporation of normal HMEC and MCF-7 cancer cells, while **CLA** was inhibitory. Supplementation with LA increased intracellular lipid peroxide concns. in normal HMEC and MCF-7 cancer cells, whereas **CLA** did not affect lipid peroxide formation. Normal HMEC and CF-7 cells supplemented with LA and INDO or NDGA resulted in growth inhibition. The treatment of normal HMEC with **CLA** and INDO or NDGA, and MCF-7 cells with **CLA** and INDO stimulated cell growth. However, the addn. of **CLA** and NDGA to MCF-7 cells resulted in synergistic growth suppression suggesting that **CLA** effects were mediated through lipoxygenase inhibition. Although NDGA was more inhibitory of cell growth in the presence of LA or **CLA** than INDO, growth was assocd. with both prostaglandin and leukotriene prodn. Addnl. studies are warranted to elucidate the mechanism(s) whereby LA or **CLA** affect breast cell growth.

IT 9029-60-1, Lipoxygenase

RL: BSU (Biological study, unclassified); BIOL (Biological study) (proliferation responses of normal human mammary and breast cancer cells to linoleic acid, **conjugated linoleic acid** and eicosanoid synthesis inhibitors)

=> d stat que

L1 302 SEA FILE=REGISTRY (LIPOXYGEN/BI OR LIPOXYGENASE/BI)
 L2 2 SEA FILE=REGISTRY "CONJUGATED LINOLEIC ACID"/CN
 L3 180 SEA FILE=REGISTRY CLA/BI
 L5 281162 SEA FILE=HCAPLUS L2 OR L3 OR CONJUGATED(W)LINOLEIC(W)ACID? OR CLA
 L10 (52)SEA FILE=REGISTRY INDOMETHACIN/BI
 L11 (1)SEA FILE=REGISTRY ETYA/CN
 L12 (36)SEA FILE=REGISTRY SHA/BI
 L13 (22)SEA FILE=REGISTRY BAICALEIN/BI
 L14 (3)SEA FILE=REGISTRY 3,4-DIHYDROXYCINNAMIC ACID?/CN
 L15 (0)SEA FILE=REGISTRY CINNAMYL(L)CYANOCINNAMATE?
 L16 (68)SEA FILE=REGISTRY GOSSYPOL/BI
 L17 (1)SEA FILE=REGISTRY "5,6-DEHYDROARACHIDONIC ACID"/CN
 L18 (0)SEA FILE=REGISTRY BACOMYCESIC ACID?/CN
 L19 (82)SEA FILE=REGISTRY 3,4-DIHYDROXYPHENYL(L)ETHANOL?
 L20 (180)SEA FILE=REGISTRY CLA/BI
 L21 388823 SEA FILE=HCAPLUS L10 OR L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR L19 OR L20
 L22 3958 SEA FILE=HCAPLUS L1 (L) (INHIB? OR REDUC? OR ACTIV? OR ADIPO? OR OBES? OR BODY(W)FAT OR FAT(W)BODY)
 L23 384 SEA FILE=HCAPLUS L21 AND L22

L44 7 SEA FILE=HCAPLUS L23 AND L5
L48 82 SEA FILE=REGISTRY 3(W)4(W)DIHYDROXYPHENYL(L)ETHANOL
L50 3 SEA FILE=REGISTRY "DOCOSAHEXAENOIC ACID"/CN
L51 1 SEA FILE=REGISTRY "EICOSATRIYNOIC ACID"/CN
L52 2 SEA FILE=REGISTRY 5-HETE/CN
L53 52101 SEA FILE=REGISTRY LACTONE/BI
L54 1 SEA FILE=REGISTRY "5(S)-HPETE"/CN
L55 1 SEA FILE=REGISTRY "12(S)-HPETE"/CN
L56 1 SEA FILE=REGISTRY "15(S)-HPETE"/CN
L58 15 SEA FILE=REGISTRY 9,12-OCTADECADIYNOIC ACID?/CN
L59 553 SEA FILE=REGISTRY PENTYL(L) (2(W)QUINOLINYLMETHOXY?)
L60 51 SEA FILE=REGISTRY TRIFLUOROMETHYL(L) PHENYL(L) 2(W)PYRAZ?
L61 1 SEA FILE=REGISTRY EPOXY(L) PHENYLIMINO(L) PROSTAGLAND?
L62 1 SEA FILE=REGISTRY NDGA/CN
L63 1 SEA FILE=REGISTRY "NORDIHYDROGUAIARETIC ACID"/CN
L64 12 SEA FILE=REGISTRY BHA/BI
L65 14 SEA FILE=REGISTRY BHT/BI
L66 88266 SEA FILE=HCAPLUS L48 OR 3(W)4(W)DIHYDROXYPHENYL(L)ETHANOL
L67 10989 SEA FILE=HCAPLUS L50 OR DOCOSAHEXAENOIC(W)ACID?
L68 82 SEA FILE=HCAPLUS L51 OR EICOSATRIYNOIC(W)ACID?
L69 1420 SEA FILE=HCAPLUS L52 OR 5(W)HETE
L70 215361 SEA FILE=HCAPLUS L53 OR LACTONE
L71 10 SEA FILE=HCAPLUS L69(L)L70
L72 140 SEA FILE=HCAPLUS L54 OR 5(W)S(W)HPETE
L73 130 SEA FILE=HCAPLUS L55 OR 12(W)S(W)HPETE
L74 244 SEA FILE=HCAPLUS L56 OR 15(W)S(W)HPETE
L76 48 SEA FILE=HCAPLUS L58 OR 9(W)12(W)OCTADECADIYNOIC(W)ACID
L77 165 SEA FILE=HCAPLUS L59 OR PENTYL(L) (2(W)QUINOLINYLMETHOXY?)
L78 443 SEA FILE=HCAPLUS L60 OR TRIFLUOROMETHYL(L) PHENYL(L) 2(W)PYRAZ?
L79 66 SEA FILE=HCAPLUS L61 OR EPOXY(L) PHENYLIMINO(L) PROSTAGLAND?
L80 3048 SEA FILE=HCAPLUS L62 OR L63 OR NDGA OR NORDIHYDROGUAIARETIC(W)A
CID
L81 5487 SEA FILE=HCAPLUS L64 OR BHA
L82 15413 SEA FILE=HCAPLUS L65 OR BHT
L83 122090 SEA FILE=HCAPLUS L66 OR L67 OR L68 OR L69 OR L71 OR L72 OR L73
OR L74 OR L76 OR L77 OR L78 OR L79 OR L80 OR L81 OR L82
L84 850 SEA FILE=HCAPLUS L83 AND L22
L85 6 SEA FILE=HCAPLUS L84 AND L5
L86 2 SEA FILE=HCAPLUS L85 NOT L44

=> d ibib abs hitrn 186 1-2

L86 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:198591 HCAPLUS
DOCUMENT NUMBER: 126:258607
TITLE: Proliferation responses of normal human mammary and
MCF-7 breast cancer cells to linoleic acid,
conjugated linoleic acid
and eicosanoid synthesis inhibitors in culture
AUTHOR(S): Cunningham, Diane C.; Harrison, Lisa Y.; Shultz, Terry
D.
CORPORATE SOURCE: Dep. Food Sci. Human Nutrition, Washington State
Univ., Pullman, WA, 99164-6376, USA
SOURCE: Anticancer Research (1997), 17(1A), 197-203
CODEN: ANTRD4; ISSN: 0250-7005
PUBLISHER: Anticancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Potential mechanisms for the stimulation or inhibition of cell growth by linoleic acid (LA) and **conjugated linoleic acid (CLA)** were investigated by using eicosanoid synthesis inhibitors. Normal human mammary epithelial cells (HMEC) and MCF-7 breast cancer cells were incubated in serum-free medium supplemented with LA or **CLA** and cyclooxygenase (indomethacin; INDO) or lipoxygenase (nondihydroguaiaritic acid; **NDGA**) inhibitors. Linoleic acid stimulated the growth and [3H]thymidine incorporation of normal HMEC and MCF-7 cancer cells, while **CLA** was inhibitory. Supplementation with LA increased intracellular lipid peroxide concns. in normal HMEC and MCF-7 cancer cells, whereas **CLA** did not affect lipid peroxide formation. Normal HMEC and CF-7 cells supplemented with LA and INDO or **NDGA** resulted in growth inhibition. The treatment of normal HMEC with **CLA** and INDO or **NDGA**, and MCF-7 cells with **CLA** and INDO stimulated cell growth. However, the addn. of **CLA** and **NDGA** to MCF-7 cells resulted in synergistic growth suppression suggesting that **CLA** effects were mediated through lipoxygenase inhibition. Although **NDGA** was more inhibitory of cell growth in the presence of LA or **CLA** than INDO, growth was assocd. with both prostaglandin and leukotriene prodn. Addnl. studies are warranted to elucidate the mechanism(s) whereby LA or **CLA** affect breast cell growth.

IT 9029-60-1, Lipoxygenase

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(proliferation responses of normal human mammary and breast cancer cells to linoleic acid, **conjugated linoleic acid** and eicosanoid synthesis **inhibitors**)

L86 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1967:470708 HCAPLUS

DOCUMENT NUMBER: 67:70708

TITLE: Inactivation of lipoxygenase by hydrogen peroxide, cysteine, and some other reagents

AUTHOR(S): Mitsuda, Hisateru; Yasumoto, Kyoden; Yamamoto, Aijiro

CORPORATE SOURCE: Kyoto Univ., Kyoto, Japan

SOURCE: Agricultural and Biological Chemistry (1967), 31(7), 853-60

CODEN: ABCHA6; ISSN: 0002-1369

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Polarography of a reaction mixt. contg. the antioxidant **nordihydroguaiaretic acid**, linoleic acid, and soybean lipoxygenase (EC 1.13.1.13) showed that H₂O₂ was not formed during the catalasecoupled oxidn. of the antioxidant. The addn. of 10-4M H₂O₂ abolished enzymic activity, and this inhibition was not reversed by cysteine or ascorbic acid. In fact, cysteine inhibited activity, 7 .times. 10-6M cysteine decreasing lipoxygenase activity by 50%. Inhibition by cysteine was lowered by the addn. of catalase or by anaerobic conditions. Since moderate oxidizing reagents such as ferricyanide did not reverse the inhibition by cysteine, it is likely that lipoxygenase was inactivated by the autoxidn. of SH groups. Inactivation by cysteine and H₂O₂ was additive and was impeded by competitive inhibitors such as linolelaidic acid and **conjugated linoleic acid**, showing a possible reaction with an amino acid residue involved in enzymic catalysis.

IT 9029-60-1, Oxygenases, lip-

(inhibition by cysteine, and hydrogen peroxide)